## **Abstract View**

## OPTICAL BRAIN MONITORING OF CEREBROVASCULAR AND INTRACELLULAR CALCIUM EFFECTS INDUCED BY ACUTE COCAINE EXPOSURE IN THE LIVING RAT

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Objective: To understand the cerebrovascular effects and cellular mechanisms caused by acute cocaine exposure using a novel multi-parametric physiological technique with milli-second temporal resolution. Using optical diffusion and fluorescence spectroscopy we assess the direct effects of cocaine on tissue blood flow, cerebral oxygenation and intracellular calcium in the cocaine-naive living rat.

Method: Six anesthetized and mechanically ventilated female rats were injected intravenously with 1 mg/kg cocaine through the left femoral vein. Cerebral blood volume, oxygenation and intracellular calcium (labeled by the calcium indicator Rhod2) were simultaneously detected from the cortical surface of the brain by optical diffusion and fluorescence spectroscopy. Standard physiological parameters, including ECG, respiration rate, arterial pressure, CO2 and body temperature were monitored during the experiments.

Results: Cocaine induced a 4.1 % decrease in cerebral blood volume and a 3.1% decrease in tissue oxygenation 3-4 min after the cocaine administration when compared to the pre-cocaine-treatment values. In parallel with these changes we observed slight decreases in the mean arterial pressure and heart rate which however remained within auto-regulatory thresholds. Interestingly, intracellular calcium transients were stable until 8.5-min after the cocaine administration, slowly increased and reached a maximal change of 26.4 6.0% at 30 min. Our findings suggest that cocaine causes cerebral vasospasm which may lead to borderline tissue ischemia as evidenced by the intracellular calcium elevation, which could be a potential contributor to development of clinical stroke observed in human subjects addicted to cocaine.

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